Hepatic granulomata may occur in primary biliary cirrhosis (Stanley *et al.* 1972) and sarcoidosis may predispose to chronic liver disease (Maddrey *et al.* 1970). Both mitochondrial and smooth muscle antibodies were negative here.

Considering his severe complications and the extensive organ involvement, this patient's clinical condition is very satisfactory though the ultimate prognosis is at best uncertain.

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Dr D M Krikler (*The Prince of Wales's Hospital, London N15 4AW*) said that on two occasions medical problems arose because of the patient's dietary habits: the more serious one of worsening hypercalcæmia when he took supplements containing calcium and vitamin D, and the carotinæmia, which was important because of possible confusion with jaundice, with consequent erroneous deduction of worse liver function. The association of ketosis with his steroid-induced diabetes could not be explained, and it was an extremely unusual occurrence.

Dr A J Karlish (*Battle Hospital, Reading*) said that sarcoid granulomata were found on routine liver biopsy in over half of cases of active sarcoidosis, and were not usually associated with jaundice or liver dysfunction. Chronic hepatitis in association with sarcoidosis was thought to have an autoimmune mechanism, as seemed to be the case in 3 patients seen in the sarcoidosis clinic in Reading. All 3 were middle aged; 2 were women. In all 3 cases sarcoidosis and liver disease were discovered simultaneously. One of the two female patients had biliary cirrhosis (Karlish *et al.* 1969), another had active chronic hepatitis, and the third had recurrent jaundice with an ill-defined form of chronic hepatitis. Such cases were more common in American negroes but rare in this country. Out of 500 patients with sarcoidosis attending a Baltimore clinic 20 had various forms of hepatitis (Maddrey *et al.* 1970). The 3 patients seen at Reading formed part of a series of 450 cases of sarcoidosis studied in the last fifteen years.

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Colitis Cystica Superficialis

M W Morris MB (for A P Wyatt FRCS)
(*Brook General Hospital, Shooters Hill, London SE18*)

Mr C R, aged 78

History: Admitted to hospital soon after returning from a holiday in Tunisia where he had developed an infected gravitational ulcer. He was treated conservatively with bed rest and given tetracycline. The ulcer healed slowly and he remained well and cheerful until he suddenly developed severe diarrhœa, passing more than 2 litres of pure mucus on some days. There was no blood in the fæces.

On examination: He was extremely ill, dehydrated and feverish. Abdomen not tender, but slightly distended. On rectal examination an extraordinarily coarse granular mucosa was felt. Biopsies of the lesions were taken through a sigmoidoscope and colonoscopy was performed. The lesions appeared as raised white nodules 2–3 mm across, scattered profusely throughout the rectum and sigmoid with normal mucosa between (Fig 1).

Investigations: Hæmoglobin fell from 14.8 to 11.3 g/100 ml. WBC 24 000/mm^3. Electrolytes: Na 124, Cl 94, K 4.1 mEq/l. Total protein 4.4 g/100 ml (albumin 2.0). Other liver function tests normal. Vitamin B_{12} 350 pg/ml. No pathogens cultured from fæces.

Barium enema showed multiple small filling defects resembling pseudopolyposis extending from rectum to cæcum (Fig 2). Barium follow-through showed the small bowel to be grossly abnormal, with coarse jejunal mucosal patterning and evidence of clumping and dilatation of lower loops of ileum. Stomach and duodenum appeared normal on gastroscopy.

Histology: Diffuse superficial cystic mucosal change, the cysts being superficial to the muscularis mucosa, and filled with mucoid material and some inflammatory cells (Fig 3).

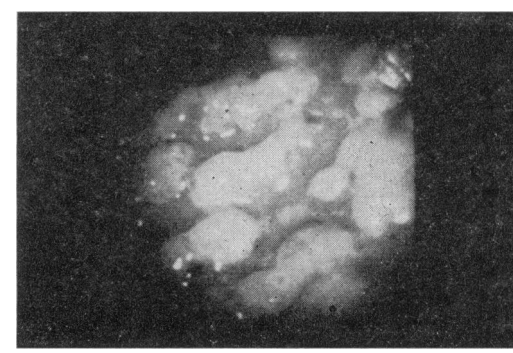

Fig 1 *Colonoscopy appearance of rectal mucosal lesions*

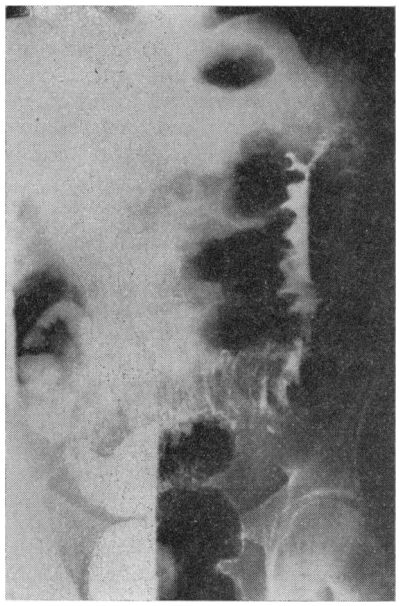

Fig 2 *Barium enema showing polyposis throughout*

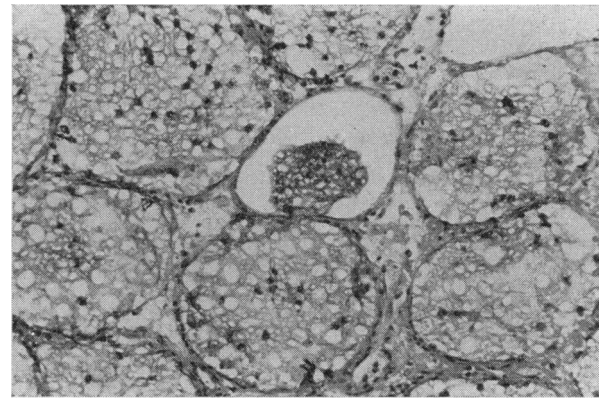

Fig 3 *Histology of rectal biopsy showing colitis cystica superficialis. Preparation stained with PAS to demonstrate mucopolysaccharides*

Treatment and progress: The patient was treated medically as for severe ulcerative colitis. There was no improvement after a week on local steroids so large doses of systemic steroids were given and sulphasalazine added. The diarrhœa ceased about a week after starting systemic steroids and the patient recovered completely. Recent sigmoidoscopy and rectal biopsy showed a return to complete normality of the rectal mucosa.

Discussion

The diagnosis of colitis cystica superficialis is essentially a histological one characterized by the development of dilated epithelial tubules giving a cystic appearance to the mucosa (Goligher & de Dombal 1968). The condition is quite distinct from the colitis cystica profunda found in chronic ulcerative colitis, where epithelium is displaced into the submucosa causing the development of numerous cysts (Goodall & Sinclair 1957, Epstein 1966). Recently it was pointed out (Morson 1972) that there is characteristically a depletion of goblet cells in ulcerative colitis.

The histological picture of colitis superficialis has been well described in gross vitamin deficiencies and in tropical sprue. It has also been seen in children dying of leukæmia, in some cases of thyrotoxicosis, in uræmia and in mercury poisoning (Wright & Symmers 1966).

In a masterly account of the pathology of pellagra, Denton (1925) described large bowel pathology very similar to colitis cystica superficialis. Small grey cystic bodies filled with pus and mucus very much like those illustrated in Fig 1 were also found.

Histology has been described in two cases (Cronkhite & Canada 1955) presenting with alopecia, hyperpigmentation of the hands and arms, atrophy of the nails and extensive gastrointestinal polyposis, where there was evidence of superficial mucosal cystic change. Similar cases have since been described (Johnston *et al.* 1962, Jarnun 1966). Ryall (1966) reported a case of polypoid hypertrophy of the gastrointestinal tract presenting as ulcerative colitis. Colitis cystica superficialis was diagnosed histologically, and the case was treated as fulminant ulcerative colitis; the patient died a few days after operation, and extensive small bowel involvement was found at post-mortem.

In seeking a cause for this very rare condition gross vitamin deficiency was excluded, thyroid function tests were normal, there was no evidence of uræmia and there seemed to be no infective cause, as repeated stool cultures were negative.

It would appear to be important to separate this condition clearly from ulcerative colitis for, in view of the small bowel involvement and the fatal outcome of surgically treated cases, ablative colonic surgery is strongly contraindicated.

Acknowledgments: Thanks are due to Dr I G Williams for Fig 3 and to Dr C N Mallinson for Fig 2.

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Mr A G Cox (*Northwick Park Hospital, Harrow*) said he did not understand the pathology of the lesion seen in the colon. He wondered whether it was a non-specific reaction of the colon to various stimuli which in this case had flared up in response to the tetracycline.

Dr P S Burge (*Whittington Hospital, Archway Wing, London N19*) thought it surprising that the patient did not develop hypokalæmia in view of the profuse mucous diarrhœa.

Mr J S Kirkham said that within the last year and a half he had seen two patients, an Indian woman in Kenya and an Englishman in London, who had both developed fulminating ulcerative colitis following a course of tetracycline. Both patients were in their third decade and the woman was in the puerperium.

Mr A P Wyatt said that the message from this case was that surgery was definitely contraindicated in this condition. There was a great temptation for the surgeon to offer the patient a total proctocolectomy as he was so ill with diarrhœa; but a study of the literature led to radiological examination of the small bowel and confirmed the decision to use medical measures only. The patient required intravenous replacement of lost fluids but the attack appeared to be aborted by a high dose of steroids (prednisone 30 mg eight-hourly) which was reduced rapidly.

Rheumatoid Arthritis and Systemic Lupus Erythematosus (SLE)

T C Beer MB MRCP (for J M Gumpel BM MRCP)
(*Northwick Park Hospital, Harrow, Middlesex*)

Mrs N B, aged 34. Housewife

History: Developed classical rheumatoid arthritis in 1959, aged 20. There was a marked erosive arthritis, subcutaneous nodules and a highly positive rheumatoid factor test (titre 1:1280). No systemic complications occurred.

In June 1972 there was an abrupt change with the development of a widespread erythematous rash. This was painful rather than pruritic and associated with intermittent fever, increasing joint pains and general ill health. Vasculitis of the fingers developed, purpura over the shins, and alopecia. Other evidence of the change to a systemic illness were three episodes of severe, but unexplained, abdominal pain; one was associated with a pleural effusion.

Investigations: Anti-nuclear factor negative on 14 July 1972, but became positive on 26 July (titre 1:250) and has remained so in significant titres. LE cells were seen in most microscopic fields. Complement levels were low on 14 July (total hæmolytic complement 10_{CH50} units, C3 18 mg/100 ml) but steadily rose to normal ($>35_{CH50}$ units, C3 >120 mg/100 ml) by October 1972. Serum anti DNA antibodies were measured (Dr Gerald Johnson) using the Farr technique (Farr 1958, Wold *et al.* 1968). DNA binding capacity values in excess of 10% are taken as indicative of SLE, and between July 1972 and January 1973 values ranging from 21.2%–18.4% were obtained. There was no direct evidence of renal involvement.

Discussion

Reports of associations between RA and SLE were recently reviewed by Kantor *et al.* (1969); few of the 116 reports adequately established both diagnoses.

There is no doubt that this patient developed classical RA in 1959. The subsequent systemic illness could have been (1) phenylbutazone-induced SLE, (2) an example of the 20% of RA which have positive antinuclear factor tests, or (3) the development of SLE in addition to RA. There was not a close relationship between phenylbutazone intake and the rash, and anti DNA antibodies to native DNA are not found in drug-induced SLE or rheumatoid disease alone.

The diagnosis of SLE in addition to RA was therefore made and steroid therapy was begun in September 1972, with good effect.

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Successful Repair of Acute Traumatic Left Subclavian Artery Steal Syndrome

Kenneth G Reid[1] MB FRCS
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R C, man aged 21. Wood machinist

History: This patient was thrown from a motor vehicle following a collision. On admission to a casualty department he was unconscious, with no

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